Gastric ulceration in an equine neonate

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Abstract — A 24-hour-old colt presented with clinical signs consistent with gastric ulceration. Treatment was initiated with a histamine type-2 receptor antagonist and clinical signs resolved. Gastroscopy at 16 d confirmed the presence of a gastric ulcer. Although gastric ulceration is common in foals, it is rarely reported in foals this young.

Résumé — Ulcération gastrique chez un poulain nouveau-né. Un poulain âgé a 24 heures présente des signes cliniques compatibles avec une ulcération gastrique. Un traitement a été entrepris avec un antagoniste des récepteurs H_2 de l'histamine, et les symptômes ont disparu. Une gastroscopie au jour 16 a confirmé la présence d'un ulcère gastrique. Bien que l'ulcération gastrique soit répandue chez les poulains, le cas est rarement signalé chez des poulains aussi jeunes.

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A 24-hour-old colt was presented with a history of colic since birth. Clinical signs included rolling and lying in dorsal recumbency. The 1st episode occurred after the first ingestion of mare's milk and resolved between feedings, recurring after nursing. The foaling, attended by the owners, was reported to be normal. Gestational age of the foal was 320 d, and there were no signs of dysmaturity. Meconium was passed without difficulty.

On physical examination, the foal's heart and respiratory rates were slightly elevated. All other parameters were within normal limits. The foal had umbilical and bilateral inguinal hernias that were totally reducible and, therefore, deemed not to be the cause of the colic. Blood was collected for a complete blood cell (CBC) count and plasma fibrinogen determination (QBC VetAutoread Hematology System; Idexx, Westbrook, Maine, USA) and immunoglobulin (Ig)G levels (SNAP Foal IgG test kit, Idexx). All results were within normal limits. On the basis of the history and findings on the clinical examination, a tentative diagnosis of gastric ulceration was made. Differential diagnoses included atresia coli, meconium impaction, ileus, colitis, and ruptured bladder.

The foal was treated with oral cimetidine (Apo-Cimetidine; Apotex, Toronto, Ontario), 25 mg/kg BW, PO, q8h. Within 48 h, the foal had improved, and by 72 h, it was clinically normal. At 16 d of age, the foal was again examined to evaluate its progress. In the interval, there had been no further signs of colic, and the umbilical and left inguinal hernias had resolved. Endoscopic examination of the gastric mucosa was performed after sedation with a combination of xylazine hydrochloride (Rompun 100 mg/mL; Bayer, Etobicoke, Ontario), 0.5 mg/kg BW, and butorphanol tartrate (Torbugesic; Ayerst, Guelph, Ontario), 0.01 mg/kg BW, both administered IV. Feeding was not restricted prior to endoscopy because the foal was so young.

There was a 10- × 15-cm, healing ulcer in the squamous mucosa of the stomach, with occasional adherent fibrin tags. The remainder of the mucosa appeared normal. The length of the endoscope was insufficient to examine the duodenum. Medication was changed to omeprazole (Omeprazole; Veterinary Pharmacy, Guelph, Ontario), 4 mg/kg BW, PO, q24h, to further aid in healing of the ulcer. Three months after initial presentation, the colt showed no signs of gastric ulceration and required no further treatment.

Gastric ulceration is common in the equine species. It is most commonly diagnosed in compromised foals and performance horses, and is referred to as gastroduodenal ulcer syndrome. The reported prevalence of ulcers in foals is 25% to 57% (3). Retrospective studies show that gastric ulcers have not been found in aborted fetuses, indicating that gastric ulcers do not commonly occur before birth, nor have they been reported in foals that have died due to dystocia. The majority of gastric ulcers in foals are reported in animals more than 2 d of age (2). The most common clinical signs include anorexia, bruxism, pytalism, dorsal recumbency, and colic (2–5). The colt in this case showed only dorsal recumbency, but the presenting signs of gastric ulcers do vary and some ulcers may not be evident clinically. Foals may die suddenly due to gastric or duodenal perforation without prior signs that suggest ulceration. Diarrhea often occurs in foals showing clinical signs (2,4). Ulcers are not commonly described in apparently healthy neonatal foals, but as no studies have been performed to identify the problem, this may not be a reflection of the prevalence of gastric ulceration in neonates.

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There are a variety of presumed causes of gastric ulceration in foals. These include physiologic stress, hypoxia, delayed gastric emptying, prolonged time between feedings, small meal size, and prolonged recumbency (2). Nonsteroidal antiinflammatory drugs (NSAIDs) are a common cause of gastric ulceration because of their inhibitory effects on the production of protective prostaglandins (4,6). Illness increases the risk of ulceration by decreasing gastric mucosal defenses. The exact mechanism for this is not clear, but decreased blood flow to the gastric mucosa is thought to play a role (7).

In this case, none of the described causative factors were identified. The foal appeared healthy at birth and nursed within a normal interval. There was no history of the mare being treated with NSAIDs during gestation; nor had the colt been medicated. There may have been a period of hypoxia during parturition that was not perceived by the owners, but clinical signs of this were not evident. So, it is possible that, in this case, some other source of abdominal pain at birth resulted in gastric ulceration.

Foals have a gastric pH of $>$ 4.0 at birth, but this decreases with age, and by 1 wk of age, gastric pH is often $<$ 2.0. Nursing causes an abrupt increase in gastric pH in 2 ways. Ingestion of milk helps to stimulate secretion of saliva, which is alkaline and increases gastric pH. In addition, milk in the stomach absorbs acidic gastric secretions (2). With increased time between feedings, gastric fluid remains highly acidic (2,3). Frequent nursing or feeding is therefore beneficial in preventing formation of gastric ulcers and should be considered when dealing with sick foals in situations that put them at greater risk, such as being recumbent or anorexic for prolonged periods. This colt's appetite remained good, and it was not deemed necessary to supplement his diet. The owners were instructed to monitor feed intake closely, to minimize the risk of ulceration.

Definitive diagnosis of gastric ulceration in foals can only be achieved with gastroscopy (2,4,5). Radiography and a barium series may help to detect delayed gastric emptying or duodenal stenosis caused by ulceration. In severe cases, gastric ultrasonography and abdominal paracentesis may also be beneficial in diagnosis (2). If diagnostic imaging is not available, the benefits of empirical treatment with anti-ulcer medication should be considered. Medical treatment is usually required, although mild lesions may heal without treatment. The goal of treatment is to suppress gastric acid secretion by using histamine type-2 receptor antagonists, such as cimetidine or ranitidine, or a proton pump blocker, such as omeprazole. Foals treated with inadequate dosage of histamine type-2 receptor antagonists may show clinical improvement without complete healing of ulcers. Antacids may also be useful for treatment, but they have a short duration of action. Sucralfate (sucrose octasulphate) may be administered in conjunction with acid inhibitors. Sucralfate binds to the ulcerated mucosa, inhibiting pepsin digestion of the mucosa, and enhances the mucus-bicarbonate gastric mucosal barrier, local prostaglandin production, and blood flow to the gastric mucosa. In cases of severe ulceration, or if the duodenum is involved, drugs that enhance gastric motility may aid in removing acidic secretions and in delivering medication to the small intestine. Bethanacol, a cholinergic agonist, is the drug of choice (2).

Acid inhibitors alone were deemed sufficient treatment for the colt in this case, because the clinical signs were mild and there was a rapid clinical response to cimetidine. Possible reasons for the failure of cimetidine treatment to allow complete healing of the ulcer in this case include owner noncompliance, under-dosing, or difficulty in medicating the foal. Omeprazole, which was avoided initially because of its expense, was prescribed because of its better efficacy (1) and because once-a-day dosing was easier for the owner.

Prevention of gastric ulceration in foals is difficult. A foal that is subjected to physiological stresses or prolonged periods of recumbency, or that is being transported long distances with its dam, should be considered at risk. It is important to ensure that foals are nursing or being fed frequently. Prophylactic treatment with a histamine type-2 receptor antagonist or proton pump inhibitor should be initiated in foals at risk, even without signs of disease, as it is possible for a gastric ulcer to perforate with no previous clinical signs (2,3).

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